How to prevent cannabis-induced psychological distress . . . in politicians

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Cannabis can cause anxiety, agitation, and anger among politicians. The consequences of this cannabis-induced psychological distress syndrome (CIPDS) include over-reaction with respect to legislation and politics and a lack of distinction between use and misuse of cannabis. In times of a war against drugs, this distinction might even be regarded as unpatriotic,1 as irresoluteness in the face of the enemy. One trend associated with CIPDS involves taking away the driving licence of people who drive and are discovered to have inactive tetrahydrocannabinol metabolites in their urine.2 In a more severe state of paranoia even medicinal use can be perceived as a threat to society, since it might “destabilize the societal norm that drug use is dangerous”,3 ignoring the fact that many prescription and over-the-counter drugs are potentially harmful. Exaggerated laws on cannabis made by anxious individuals could be regarded as a modern version of the generational conflict.4

Rationality and factuality are needed to calm down politicians affected by CIPDS. That cannabis might cause infidelity, cancer, cognitive decline, dependency, traffic accidents, and heart attacks, and that it can lead to the use of more dangerous drugs, are all arguments encountered when studying drug use, such as the limits of confounder adjustment. The results of one often-cited Swedish study,5 for example, indicate a crude odds ratio of 6·7 for schizophrenia risk at age 26 years in individuals who used cannabis more than 50 times before age 18 years. This finding suggests cannabis is an important contributor to schizophrenia. After adjustment for several possible confounders, however, the risk decreased to 3·1, a strong indication of residual confounding—ie, the presence of factors that would further reduce the risk if included in the statistical model but that could not be included because of a lack of data.

Another review6 details the findings of an investigation into the association between cannabis and psychosis on the basis of five longitudinal studies. The authors conceded that only one of these studies was able to record whether prodromal manifestations of schizophrenia preceded cannabis use. The results of the study indicated that “cannabis users at age 18 years had elevated scores on the schizophrenic symptom scale only if they had reported psychotic symptoms at 11 years”, and that people who used cannabis at age 15 years had a higher risk for adult schizophrenia disorder at age 26 years even if psychotic symptoms at age 11 years were controlled for.7 The researchers concluded that cannabis was a causal factor for psychosis in “vulnerable youths”.8

There is some reason to believe that cannabis contributes to psychosocial problems in adolescents and young adults, and no responsible adult would want young people to take drugs. There is no question that this issue is an important candidate for education and prevention, but there is a fierce debate on the place repressive measures should have in this context. There is little reason to believe that criminalisation has had a strong effect on the extent of cannabis use by young people.9 Moreover, prohibition itself seems to increase the harmfulness of drug use and cause social harm.

By stopping all cannabis users from being treated as criminals, I believe this year’s change by the British Government of its cannabis law (a declassification from class B to C) is a sensible attempt to balance the possible harms caused by cannabis and its prohibition. The concern expressed by Peter Maguire of the British Medical Association and others,10 that “the public might think that reclassification equals safe”, is based on the wrong assumption that cannabis became illegal because its use is unsafe and dangerous. Many unsafe activities are legal, including skiing downhill, having sex, drinking beer, eating hamburgers, and taking aspirin. Cannabis did not become illegal because it was shown to be dangerous but, more likely, because Harry Anslinger, Commissioner of the US Bureau of Narcotics 1930–62, and his colleagues needed a new target and battlefield after the end of alcohol prohibition in 1933. Reputed dangers, presented in his statements before the US Senate in 1937,12 were used as a shocking means of manipulation—eg, “A man under the influence of marijuana actually decapitated his best friend; and then, coming out of the effects of the drug, was as horrified as anyone over what he had done.” The representative of the American Medical Association strongly opposed the Marijuana Tax Act of 1937: “To say . . . that the use of the drug should be prevented by a prohibitive tax, loses sight of the fact that future investigation may show that there are substantial medical uses for cannabis.”13

We live in a time in which the unrealistic and unproductive paradigm of complete abstinence from drugs is slowly dissipating. Proponents of a drug-free society find this fact hard to accept, and responsible politicians and doctors can find achieving an appropriate position in the debate difficult. However, we must learn to deal with drugs and their possible dangers without fear.

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Psychological, physiological, and drug interventions for type 2 diabetes

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Type 2 diabetes is perhaps one of the best recognised lifestyle-associated diseases. The number affected is expected to double over the next 20 years as more people suffer the consequences of greater availability of food and less requirement for exercise. To many, the contemporary man-made environment appears so inhospitable that drug interventions are increasingly required to preserve good health. Yet sustained application of diet and lifestyle change even in the face of a hostile environment can reduce the development of diabetes even more successfully than prophylactic drug therapy (metformin), as shown in the Diabetes Prevention Trial.1 The problem is how to help psychologically challenged (stressed) people deal with the strains of life and enable effective compliance with the hygienic principles of dietary restraint and increased exercise. For those who already have type 2 diabetes, further stresses are added, such as self-monitoring of blood glucose, taking oral hypoglycaemic agents, and increasingly insulin injections, and foot care. It is therefore of particular interest in this issue of The Lancet that Khalida Ismail and colleagues report the results of a systematic review and meta-analysis of randomised controlled trials of psychological interventions to improve glycaemic control in patients with type 2 diabetes.

Ismail and colleagues searched four databases in 2003 (MEDLINE, PsyCINFO, EMBASE and the Cochrane Central Register of Controlled Trials) for 2427 abstracts, leading to a meta-analysis of twelve trials that included measurement of glyced haemoglobin as an outcome, eight for blood sugar, nine for bodyweight, and five for psychological status. Surprisingly, there was a reduction in glyced haemoglobin of 0·76 in absolute units not dissimilar to that achieved in the UKPDS trial.2 Furthermore, when two studies were excluded in which the control arm was not a true control but a less intensively applied psychological therapy, the advantage in glyced protein rose to 1·06 units. Such differences over time would be expected to greatly reduce complications. No significant reductions were seen in blood glucose, body weight, or psychological distresses. The lack of weight gain was encouraging in view of the rise in body-weight often seen with conventional intensive therapy for diabetes to achieve this level of improvement in glyced haemoglobin.3 However, there might have been hope for weight loss since the second most common form of psychological therapy, motivational interviewing, has been used in both tobacco cessation and weight-loss programmes.4

No blood-lipid data were reported in Ismail and colleagues’ study, although these would have been useful markers of increased attention to diet and lifestyle resulting from the psychological intervention. It is not obvious by what means the psychological intervention exerts its beneficial effect. Is it by closer attention to blood-glucose monitoring with changes in oral hypoglycaemic drugs or insulin dose? Is there greater compliance with dietary advice or is more exercise taken on a regular basis? These issues could usefully be followed up in the future since the psychological therapy could then be paired with the appropriate educational package to maximise the overall effect.

Regardless of mechanism, the size of the effect on glyced proteins demands serious consideration as an important therapeutic achievement likely to reduce microvascular complications and possibly even macrovascular disease. The problem lies in its application. Thus, although it is increasingly recognised, for example, that motivational interviewing is valuable, it is likely to require considerably more time with the patient and a greater frequency of visits. This scenario is the antithesis of current busy hospital clinics or doctors’ offices where at best a tightly scripted comprehensive check-list of questions and statements are applied with speedy efficiency.

Ismail and colleagues’ report suggests that the current treatment of diabetes can be significantly improved by non-pharmacological means. Will diabetes treatment be both started and followed up in groups to allow more time for discussion and will these meetings have some of the characteristics of Alcoholics Anonymous or Weight Watchers? Unless we can alter the external environment that is creating diabetes and other chronic diseases, we will have to find new ways of effectively treating the oncoming epidemic (ie, we may have to mop up rather than turn the tap off). Ismail and colleagues offer hope, but the treatment of diabetes with psychological, in addition to physiological (diet, exercise, and weight loss), and, as necessary, drug therapy, will require an investment of time and a broad range of expertise.

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